90 Heart 1996;76:90-92

LETTERS TO THE EDITOR

Scope

Heart welcomes letters commenting on papers published in the journal in the previous six months. Topics not related to papers published earlier in the journal may be introduced as a letter: letters reporting original data may be sent for peer review.

Presentation

Letters should be:

- not more than 600 words and six references in length
- typed in double spacing (fax copies and paper copy only)
- signed by all authors.

They may contain short tables or a small figure. Please send a copy of your letter on disk. Full instructions to authors appear in the July 1996 issue of *Heart* (page 93).

Serum lipids four weeks after acute infarction are a valid basis for lipid lowering intervention in patients receiving thrombolysis

SIR,—As long ago as 1971 we first suggested that patients with myocardial infarction should be screened for hyperlipidaemia on the first morning after admission.1 We were disappointed to note that Carlsson et al failed to acknowledge this,2 quoting our 1971 paper in support of the statement that "serum lipid concentrations are usually not assessed after myocardial infarction" when we in fact concluded that "hyperlipidaemic states can be identified during the twentyfour hours after a myocardial infarction".

We also showed that the magnitude of the fall in cholesterol after myocardial infarction seemed to be related to the severity of the episode. We were therefore surprised that Carlsson et al did not supply data comparing the size of infarction in their thrombolytic groups with their non-thrombolytic groups. This, not minor differences in the timing of the initial sample as they suggest, seems to us to be the most likely explanation of the differences in cholesterol that they showed between the groups.

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- 1 Fyfe T, Baxter RH, Cochran KM, Booth EM.
- Fyfe T, Baxter RH, Cochran KM, Booth EM. Plasma lipid changes after myocardial infarction. Lancet 1971;ii:997-1001.
 Carlsson R, Lindberg G, Westin L, Israelsson B. Serum lipids four weeks after acute myocardial infarction are a valid basis for lipid lowering intervention in patients receiving thrombolysis. Br Heart J 1995;74:18-20.

Circadian variation of left ventricular diastolic function in healthy people

SIR,—In their study of healthy people, Voutilainen et al found a nocturnal decrease and a daytime increase in the rate of left ventricular relaxation, which they tentatively attributed to sympathoadrenal activity.1 Neurobiological features are suggested by reports that link dysregulation of brainstem cardiovascular control and cardiovascular reactivity in challenging tasks with dopamine abnormalities lateralised to the right hemisphere. This hypothesis is supported by the importance of dopamine in the control of wakefulness manifested by a reduction of reaction time and gap frequency, optimal response organisation at intermediate dopamine tone in a medial-frontal-striatal activation system, and inhibition of the right hemisphere promoting left-hemisphere dominance associated with cardiac arrhythmia and vasoconstriction. These observations prompt a multidisciplinary approach involving neuropharmacology and cardiovascular physiology² in evaluating the pathophysiological importance of diurnal changes1 and time course of speech induced ischaemia.3 This method is supported by the correlation of the frequency and duration of speech hesitation pauses with a sixfold increase in the prevalence and incidence of coronary heart disease and mood, respectively, which reflect properties of neuronal activity and firing, and by the correlation of anxiety with the sixfold increase in fatal coronary heart disease, in particular, sudden cardiac death. It is also supported by the reduction in blood pressure associated with longer, less recurrent pauses4 that is predictive of a response to neuropharmacological intervention,5 and by the association of pause rate and variability in pause duration with the left and right hemisphere, respectively46—hence the need to tailor interventions to asymmetrical brain functions.

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Voutilainen S, Kupari M, Hippelainen M, Karppinen K, Ventila M. Circadian variation of left ventricular diastolic function in healthy people. Heart 1996;75:35-9.
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 Ahlawat SK, Legault SE. Pathophysiology and time course of silent myocardial ischaemia during mental stress (letter and reny). Heart

during mental stress (letter and reply). Heart 1996;75:101.

- 4 Friedman EH, Coyle CP, Santiago MC. Neurobiology of depressive symptoms (letter and reply). Arch Phys Med Rehabil 1995;76:
- 5 Ranelli CJ, Miller RE. Behavioral predictors of
- amitriptyline response in depression. Am J Psychiat 1981;138:30-4.

 6 Blonder LX, Pickering JE, Heath RL, Smith CD, Butler SM. Prosodic characteristics of speech pre- and post-right hemisphere stroke. Brain Lang 1995;51:318-35.

Effect of percutaneous fenestration of the atrial septum on protein-losing enteropathy after the Fontan operation

SIR,—We read with interest the paper by Mertens et al.1 They reported that in patients who have a poor clinical result after Fontan operation, fenestration of the interatrial septum should be considered before proceeding to Fontan take-down or heart transplantation.

We recently treated a boy with hypoplastic left heart syndrome who had had a Norwood operation as a neonate and hemi-Fontan operation at the age of six months. The Fontan (TCPC) operation was completed when he was one year old and he was discharged on 57th postoperative day. The postoperative course was uneventful and he was doing well at home for a month, before he returned to hospital with right heart failure. An intra-atrial venous route was created by using 10 mm Gore-tex graft. Right heart failure progressed to protein-losing enteropathy which was resistant to medical treatment and finally we decided to construct a percutaneous fenestration of the interatrial septum made with a Gore-tex graft. A 6 French long sheath was introduced into the intra-atrial venous chamber from internal carotid vein, a Brockenbrough needle was introduced through the long sheath. A Blalock-Park blade catheter and 10 mm balloon catheter were introduced and a 10 mm diameter hole was made. Low cardiac output improved immediately. Transophageal echocardiography immediately after the procedure showed a 7 mm diameter hole with significant right-to-left shunt in the Gore-tex baffle. The patient looked well and ascites and pleural effusion were reduced. However, the pleural effusion and ascites gradually returned within three weeks. Because he showed clinical signs of severe low cardiac output we decided on a Fontan take-down 41 days after the transcatheter fenestration. The patient died from multiorgan failure three days after the take-down operation. At operation we found that the fenestration in the Gore-tex graft had closed. The Gore-tex graft had thickened up to 2 mm and the hole was completely closed and covered by endocardium.

Mertens et al1 reported successful treatment of a patient whose native interatrial septum was fenestrated by means of a Brockenbrough needle. Our case suggested that early closure of the fenestrated hole in the Gore-tex baffle is likely.

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1 Mertens L, Dumoulin M, Gewillig M. Effect of percutaneous fenestration of the atrial septum on protein-losing enteropathy after the Fontan operation. Br Heart J 1994;72:591-2.

This letter was shown to the authors, who reply as follows:

SIR,—We read with interest the case described by Satomi et al in which they confirmed the effectiveness of a secondary fenestration as a treatment for protein-losing enteropathy (PLE) after Fontan operation. We share their experience of seeing a significant haemodynamic and clinical improvement in patients immediately after the fenestration was created. Also like us and experience with cases we are aware of in other centres where this technique has been applied, Satomi et al observed a strong tendency of the percutaneous fenestration to close. The three fenestrations we created in Fontan patients, all became considerably